

REFERENCES

1. Goodman, L. and Gilman, A. 1980: Pharmacologic Basis of Therapeutics, editors of Sixth Edition; Macmillan Pub. Co., U.S.A.
2. Brimblecombe, R. W. 1974: Drug Actions on Cholinergic Systems. University Park Press, Baltimore.
3. Weiner, N. Neurotransmitter systems in the central nervous system. In Drugs and the Developing Brain. (Vernadakis, A., and Weiner, N., eds.) Plenum Press, New York, 1974, pp. 105-131.
4. Rumack, B. H. 1973: Anticholinergic poisoning: treatment with physostigmine. Pediatrics, 52, 449-451.
5. Ketchum, J. S.; Sidell, F. R.; Crowell, E. G., Jr.; Aghjanian, G. K.; and Hayes, A. H., Jr. 1973: Atropine, Scopolamine, and Ditran: Comparative pharmacology and antagonists in man. Psychopharmacologia, 28, 121-145.
6. Shader, R. I., and Greenblatt, D. J. 1972: Belladonna alkaloids and synthetic anticholinergics. Uses and toxicity. In, Psychiatric Complications of Medical Drugs. (Shader, R. I., ed.) Raven Press, New York, 103-147.
7. Forrer, G. R. and Miller, J. J. 1958: Atropine coma: a somatic therapy in psychiatry, Am. J. Psychiatry, 115, 455-458.
8. Yamamura, H. I. and Snyder, S. H. 1974: Muscarinic Cholinergic Receptor Binding in the Longitudinal Muscle of the Guinea Pig Ilium with [³H] Quinuclidinyl Benzilate. Mol. Pharmacol. 10: 861-867.
9. Hulme, E. C.; Birdsall, N. J. M.; Burgen, A. S. V.; and Mehta, P. 1978: The binding of antagonists to brain muscarinic receptors. Mol. Pharmacol., 14, 737-750.
10. Levine, R. M. 1959: The intestinal absorption of the quaternary derivatives of atropine and scopolamine. Arch. Int. Pharmacodyn. Ther., 121, 146-149.
11. Jonkman, J. H. G.; Van Bork, L. E.; Wijsbeek, J.; De Zeeuw, R. A.; and Orie, N. G. M. 1977: Variations in the bioavailability of thiazinamium methylsulfate. Clin. Pharmacol. Ther., 21, 457-463.
12. Zvirblis, P. and Kondritzer, A. 1966: Adsorption of H³BZ and C¹⁴-atropine to the mitochondrial fraction of the rat brain. Edgewood Arsenal Tech. Rept. 4042.
13. Sidell, F. R. undated: A summary of the investigations in man with BZ conducted by the U.S. Army, 1960-1969. CSL 000-137.

14. Snyder, S. H., Chang, K. J., Kuhar, M. J., Yamamura, H. I. 1975: Biochemical Identification of Mammalian Muscarinic Cholinergic Receptor. Fed. Proc. 34:1915-1921.
15. Yamamura, H. I., Chang, K. J., Kuhar, M. J., Snyder, S. H. 1975: Cholinergic Muscarinic Receptor: Biochemical and Light Autoradiographic Localization in the Brain. Croat. Chem. Acta. 47:475-488.
16. Becker, M. J. 1964: Biochemical Studies of BZ and EA 3443. Final Report, Contract No. DA-18-104-CML-6631.
17. Yamamura, H. I., Kuhar, M. J., Greenberg, D., Snyder, S. H. 1974: Muscarinic Cholinergic Receptor Binding: Regional Distribution in Monkey Brain. Brain Res. 66:541-546.
18. Ketchum, J. 1963: The human assessment of BZ. Chemical Research and Development Laboratories Tech. Memo 20-29.
19. Lowy, K., Abood, L. G. and Rains, H.: 1976. Behavioral effects and binding affinities of two stereoisomeric psychotomimetic glycolates. J. Neurosci, Res. 2, 157-165.
20. Abood, L.G. and Rinaldi, F.: 1959. Studies with a tritium labeled psychotomimetic agent. Psychopharmacol. 1:119-123.
21. Shallek, W. and Smith, T. H. F. 1952: Electroencephalographic analysis of side effects of spasmolytic drugs. J. Pharmacol. Exp. therap. 104:291-298.
22. McNamara, B. P. 1960: Research in Toxicology Division on Effects of CS4030 in animals. Chemical Warfare Laboratories Special Pub. 2-28.
23. Mennear, J., Samuel, G., Jennings, H., McCarthy D., OH, J., Marshall, E. and Smith, B. Preclinical Pharmacology Study - Monkeys (EA3443). Contract DA 18-108-AMC-78[A] (C73, Index 105).
24. Mennear, J., Jennings, H., Kuldell, K., Hall, D., Poland, R. and Lamb, R. Preclinical Pharmacology Study - Dogs. Contract DA-18-108-405-CML-826.
25. Shiner, P. T., Maj., M. C., Agent EA3834-renal evaluation as of Jan. 1970, Clinic Investigation Branch, Clinical Medical Sciences Dept., Med. Res. Labs.
26. Boyd, C. E. and Boyd, E. M. 1962. The chronic toxicity of atropine administered intramuscularly to rabbits. Tox. Appl. Pharm. 4:457-467.
27. Bagdon, R.E., 1965: One year chronic toxicity studies of quartzan in dogs, Dec. 13, 1965. Hoffmann-LaRoche, Inc. (Prepared by Food and Drug Res. Labs. Inc. and submitted to Hoffmann LaRoche Dec. 8, 1965 for use in connection with new drug application to FDA for human use).

28. Bagdon, R.E. and Garner, E., 1966: One year chronic toxicity studies of clidinium bromide in rats. April 20. Hoffmann-LaRoche, Inc.
29. Carson, S. 1965: Chronic (1-year) oral dosage studies with Quartsan in dogs. Report from Food and Drug Research Labs., Inc. to Hoffmann LaRoche, Inc.
30. Sram, R. J., Kucerova, M. and Bardode, Z. 1975: Mutagenic activity of 3-chinuclidylbenzilate.
31. Crowell, E. B., Jr., and Ketchum J. S. 1967: The treatment of scopolamine-induced delirium with physostigmine. Edgewood Arsenal Tech. Rept. 4115.
32. Klapper, A. J., McCarroll, J. E. and McColloch, M. 1972: Long-term followup of medical volunteers. Edgewood Arsenal Tech. Rept. 4611.
33. Hart, J.J. and Balter, L.: 1966. A study of possible residual EFFE CTS of EA 3443 and EA 3580 on COG Nitve Ability [U]. Aberdeen Proving Ground, Maryland.
34. Abood, L.G. and Meduna, L.J.: 1958. Some effects of a new psychotogen in depressive states. J. Nerv. Ment. Dis 127, 546-554.
35. Meduna, L. J. and Abood, L. G. J. Neuropsych. 1:20-22, 1959. "Studies of a New Drug (Ditran) in Depressive States."
36. Finkelstein, B. A.: 1961. Ditran, a psychotherapeutic advance: A review of 103 cases. J. Neuropsychiat, 2, 144-148.
37. English, D. C.: 1962. Reintegration of affect and psychic emergence with Ditran. J. Neuropsychiat. 3, 304-310.

MORTALITY

METHOD OF ANALYSIS

The testing of chemical agents in human subjects at Edgewood which began in 1955 continued for more than 20 yr. During that period, the volunteers participated in an average of 3.1 tests each; 29% participated in only one test, and 2% participated in 10 or more tests. Because most of the volunteers participated in more than one test, the date of the most recent test was arbitrarily set as the date on which followup by the Medical Followup Agency of the National Research Council and the ascertainment of mortality began. Followup continued through December 31, 1980. The period of followup ranged from 4 to a maximum of 26 completed years.

The experience of 6,620 men is examined here. One-hundred were excluded from the study because of erroneous identification or because some essential datum, such as date of birth, was unknown. Among those studied, there were 222 deaths.

Deaths during followup were identified by matching the volunteers against a computerized file of veterans maintained by the Veterans' Administration (VA), which includes the date of death of every veteran for whom the VA has received a claim for a burial allowance and other veteran death benefits. Both name and service number were used for matching. If a death certificate showing the cause of death could not be found in the VA claim folder for a deceased veteran, a copy of the certificate was requested from the vital statistics office of the jurisdiction where the death occurred. The underlying cause of death was coded for analysis. The 31 deaths for which death certificates had not been received by the cutoff date for the receipt of data for analysis--March 31, 1982--have been classified as "cause of death unknown." These 31 deaths are distributed in similar proportions between the men who received potentially hazardous substances (15% of 134 deaths) and the men who did not (13% of 88 deaths).

The groups of volunteers cannot be compared for mortality solely on the basis of numbers of observed deaths. One must also take into account the number of years that each group has been followed and the ages of the men being observed. The volunteers in the early test years were about the same age as men used in tests conducted during the later years (Table 4-1). However, the men tested in the early years not only are under observation for a much longer period than men used in the most recent tests, but also have grown older and have had a longer followup. Consider two volunteers of the same age at testing, one whose followup began in 1955 and the other in 1970. The first man will have aged 15 yr by the time the second man is tested and will have been exposed to the risk of dying for an additional 15 yr at the end of followup. Thus, the proportion of volunteers who die during followup will decrease for each succeeding test group, in the absence of any drug effect.

The testing of drugs spanned a period of 21 yr. However, some chemical agents were tested earlier than others (Table 4-2). The testing of LSD and derivatives was concentrated in the early years; more than half the total number of doses had been administered by

the end of 1959. At the other extreme were the approved drugs, innocuous substances, and control substances, which were administered late in the testing period. One can therefore expect a much higher proportion of men in the LSD tests to have died than of the men who received approved drugs, innocuous chemicals, or control substances, even if LSD and its derivatives have no adverse effects on survival.

If one calculated the numbers of deaths expected among a test group of men, on the basis of their ages and the numbers of years they were followed, one would have a yardstick for assessing the significance of the number of deaths observed during followup. The ratio of observed deaths to expected deaths, the standardized mortality ratio (SMR), can be used to compare the mortality experiences of two or more groups. Expected deaths by underlying cause were calculated on the assumption that the volunteers experienced U.S. male, age-specific death rates for each calendar year of followup.

RESULTS

Because most of the volunteers participated in two or more tests, an analytic problem arises: If one of the test substances, say Substance A, produced detectable adverse effects, such effects would also tend to appear among men who received Substance B if there were many men tested with both substances A and B. Accordingly, data on men who were tested with any particular substance are shown in two nonoverlapping groups: those who received "only" that substance (whether once or more than once) and those who received "not only" that substance but other active substances as well.

Observed and expected numbers of deaths and the ratios of observed to expected deaths, the SMRs, by underlying cause of death in volunteers who were exposed to chemicals of various types, are shown in Table 4-3. As noted above, a distinction has been made between men who received only one type of chemical and those who were exposed to two or more types (the "not only" groups).

The SMR for the total experience, 0.81, indicates that observed mortality for all causes was only 81% of expectation. That reflects the requirement that a man meet minimal health standards to be accepted into the Army; the screening process results in a death rate that is lower than that of U.S. males in general, and this effect persists for many years. It is evident that men who were tested with active substances had been subjected to additional screening, so these men constituted an unusually fit group and might be expected to have unusually low mortality for a number of years. This phenomenon is identical with the "healthy-worker" effect that is seen in most studies of occupational group. Men who were exposed to two or more types of chemicals experienced lower SMRs than those exposed to only one type of compound, again because of selection, a man had to be in exceptionally good health to participate in multiple tests.

No "all causes" SMR exceeded 1 by an amount greater than can reasonably be attributed to chance. The high SMR, 1.11, representing an 11% excess of observed deaths, occurred in the "no test" group--men who were not exposed to any chemical agent,

possibly because of some health defect detected in the examination that preceded testing. The highest SMR, 1.20, occurred in the small group of men exposed only to the cholinesterase reactivators and represents an excess over expectation of less than a single death. The only other SMR to exceed unity occurred in men who were exposed to the anticholinergic chemicals only. Men who were exposed to anticholinergic chemicals plus other chemicals (which sometimes included substances to counteract the effects of the anticholinergic chemicals) had an SMR, 0.56, which is significantly below unity. However, when 212 men exposed to low single doses and 319 to high single doses were examined separately, no dose effect was apparent. Had the anticholinergic chemicals increased the risk of dying, one would have expected that increase to be larger in men with larger single exposures.

No evidence of a deferred, drug-induced mortality increase was seen when the experiences of the first 10 and remaining 16 follow-up years were examined separately. Of the 14 categories of experimental chemicals studied, only two had an SMR exceeding unity during the last 16 follow-up years. One of these (cholinesterase reactivator, only) was clearly due to small numbers, one observed death with 0.8 deaths expected, while the other, 47 observed versus 39.7 expected deaths, was seen in the 1,719 volunteers who were not exposed to any of the chemicals, the "no test" group. We can state with 95-percent certainty that the true SMR for these men during the first 10 years falls between 0.69 and 1.36, while the limits for the last 16 years are 0.83 and 1.54, respectively.

Most of the experimental chemicals were administered to too few volunteers to be examined separately for an influence on mortality. However, a few were considered of sufficient interest to justify being examined in that way (Table 4-4). The specific chemicals are grouped by type. The SMRs for these individual compounds range from a low of 0.80 (nine observed, 11.3 expected deaths) for the men tested with sarin only, to a high of 2.50 (five observed, two expected) for scopolamine only. Atropine, a therapeutic anticholinergic similar to scopolamine, had the next highest SMR, 1.76 (three observed, 1.7 expected). Of the eight deaths of men who received one or the other of these two therapeutically similar compounds, three were attributed to trauma (one accident, one suicide, and one other trauma), and one was due to cancer; the causes of the other four will not be known until the death certificates are obtained.

The SMRs become much more erratic when attention is directed to the underlying causes of death, because of the small expected number. If 0.5 deaths are expected, the SMR will be 0.0, 2.0, or 4.0, if only zero, one, or two deaths are observed.

Trauma was the underlying cause of 86 deaths, compared with 128.2 expected, for an SMR of 0.67. These volunteers had a significantly lower death rate from injury than that experienced by U.S. males in general. This tendency is seen in every category of test chemical, with two exceptions--the 570 men who received anticholinergic agents only (10 observed, 10.0 expected, SMR = 1.00) and the 607 men who received cholinesterase reactivators (11 observed, 11.0 expected, SMR = 1.00). In each instance, deaths due to homicide contributed to the excess. It would be difficult to attribute an increase in deaths due to trauma to effects of the two types of chemicals, because the excess is not seen among all

volunteers exposed to them. No excess is seen in the anticholinergic "not only" group or in the cholinesterase reactivator "only" group.

Three diseases were responsible for more deaths than expected among all volunteers: cancer of the respiratory tract, leukemia and aleukemia, and renal disease. All three renal-disease deaths were in the no-test group; the excess is clearly not a drug effect. The excess of respiratory-cancer deaths derives in part from the control and no-test groups; that suggests that the excess is general, not drug-related. Deaths due to respiratory cancer were responsible for an SMR of 1.11 in a group of World War II and Korean Conflict veterans (1). It is possible that the proportion of cigarette-smokers is higher among veterans than in the U.S. male population in general. The third disease causing more deaths than expected is leukemia and aleukemia (four observed, 2.8 expected, SMR = 1.43). One death occurred in the innocuous-chemical and-control group. Of the other three who died of leukemia or aleukemia, two had received anticholinesterase agents, two cholinesterase reactivators, and one each irritants, cannabis, and an unclassified chemical (included under total experience in Table 4-3). When the total volunteer experience is divided into the 1,984 men who did not receive potentially hazardous chemicals and the 4,636 men who did, the average annual death rates from leukemia are similar--3.4 and 4.0 per 100,000 person-yr of observation, respectively.

What is the chance that an increased risk of death will actually be detected? After choosing a level of significance at which the null hypothesis (no increased risk) will be rejected ($P = .05$, say), the power of a comparison is the probability that, in fact, the number of deaths observed will be large enough to constitute a significant excess over the number expected if there were no increased risk.

Power can be large if the relative risk is large or if the sample is sufficiently large. If the sample is small, then only fairly large relative risks will be detected with high probability. In particular, if 10 or more deaths would be expected on the null hypothesis, a relative risk of 2.0 would be detected in more than 80 percent of trials (power greater than 0.80). The same power is achieved if three deaths are expected only if the relative risk is at least 3.0, and if only two deaths are expected at U.S. population rates, the relative risk would have to be at least 4.0 to achieve a power of 0.80. When the number of men exposed to a given chemical are small enough or the condition at increased risk rare enough to produce an expected value of only 0.1 deaths, failure to demonstrate a significant increase would mean only that the relative risk is less than 30. From a statistical point of view, the experience being studied is incapable of demonstrating risks of dying increased less than three- or four-fold.

It can be concluded that, over the time span examined here, there is no evidence that volunteer participation in the testing programs had any long-term adverse effect on mortality.

Table 1

Numbers of Men Tested by Year of Birth
and Year of Beginning of Followup

<u>Year of Birth</u>	<u>1955- 1959</u>	<u>1960- 1964</u>	<u>1965- 1969</u>	<u>1970-</u>	<u>Total</u>
<u>No. Men:</u>					
Before 1920	35	17			52
1920-1924	55	22	2		79
1925-1929	118	78	1		197
1930-1934	417	139	17		573
1935-1939	491	837	77	17	1,422
1940-1944	47	978	805	69	1,899
1945-1949		31	1,191	571	1,793
1950-1954			18	516	534
After 1954			1	70	71
Total	1,163	2,102	2,112	1,243	6,620
<u>Mean Year of Birth:</u>					
	1934	1939	1945	1950	1942
<u>Mean Age at Beginning of Followup, yr:</u>					
	23.9	23.5	22.4	22.8	23.1

Table 2

Median Years in Which Categories of
Chemicals were Administered

<u>Category</u>	<u>Year</u>
Anticholinesterases	1962
Anticholinergics	1968
Cholinesterase reactivators	1968
Irritants	1967
Cannabis	1965
LSD derivatives	1959
Approved drugs	1971
Innocuous chemicals and controls	1971

Table 3

Observed and Expected Deaths among Test Subjects by Chemical Administered and Underlying Cause of Death

Underlying cause of death	Anticholinesterase ^a				Anticholinergic ^a				Cholinesterase reactivator ^a									
	Only (N = 507)		Not only (N = 958)		Only (N = 570)		Not only (N = 1,179)		Only (N = 83)		Not only (N = 607)							
	Obs.	Exp.	O/E		Obs.	Exp.	O/E		Obs.	Exp.	O/E							
All trauma ^b	7	11.7	0.60	13	19.8	0.66	10	10.0	1.00	11	20.6	0.53	1	1.4	0.71	11	11.0	1.00
Accidents	4	7.5	0.53	7	12.5	0.56	6	6.2	0.97	6	12.8	0.47	1	0.9	1.11	5	6.9	0.72
Homicide	1	1.9	0.53	3	3.1	0.97	3	1.6	1.88	2	3.2	0.63	0	0.2	0.0	4	1.8	2.22
Suicide	0	2.1	0.0	0	3.7	0.0	0	1.9	0.0	1	4.0	0.25	0	0.3	0.0	0	2.1	0.0
All diseases	13	17.4	0.75	10	21.9	0.46	5	8.1	0.62	5	14.9	0.34	1	1.1	0.91	4	10.0	0.40
Malignant neoplasms	5	3.9	1.28	5	4.8	1.04	1	1.8	0.56	3	3.3	0.91	0	0.3	0.0	3	2.2	1.36
Respiratory tract	1	1.1	0.91	2	1.2	1.67	0	0.4	0.0	2	0.6	3.33	0	0.1	0.0	1	0.5	2.00
Leukemia, aleukemia	0	0.3	0.0	2	0.4	5.00	0	0.2	0.0	0	0.4	0.0	0	0.0	--	2	0.2	10.00
Diseases of cardiovascular system	6	7.3	0.82	4	8.4	0.48	3	2.8	1.07	2	4.8	0.42	0	0.4	0.0	1	3.6	0.28
Chronic nephritis, other renal	0	0.2	0.0	0	0.3	0.0	0	0.1	0.0	0	0.2	0.0	0	0.0	--	0	0.1	0.0
Cirrhosis of the liver	1	1.3	0.77	0	1.7	0.0	1	0.6	1.67	0	1.2	0.0	0	0.1	0.0	0	0.8	0.0
Total deaths, all causes ^c	21	29.2	0.72	28	41.6	0.67	19	18.0	1.06	20	35.5	0.56	3	2.5	1.20	17	21.0	0.81

Table 3 (contd.)

Underlying cause of death	Irritant ^a				Cannabis				LSD derivative ^a				Drugs ^a			
	Only (N = 855)		Not only (N = 1,046)		Total (N = 252)		Only (N = 103)		Only (N = 159)							
	Obs.	Exp.	O/E	Obs.	Exp.	O/E	Obs.	Exp.	Obs.	Exp.	O/E	Obs.	Exp.	O/E	Obs.	Exp.
All trauma ^b	8	16.0	0.50	10	19.6	0.51	1	5.1	0.20	0	2.5	0.0	0	2.4	0.0	0.0
Accidents	5	10.0	0.50	7	12.2	0.57	1	3.2	0.31	0	1.6	0.0	0	1.5	0.0	0.0
Homicide	1	2.5	0.40	1	3.1	0.32	0	0.8	0.0	0	0.4	0.0	0	0.4	0.0	0.0
Suicide	1	3.0	0.33	2	3.8	0.53	0	1.0	0.0	0	0.4	0.0	0	0.5	0.0	0.0
All diseases	7	15.0	0.47	9	17.6	0.51	2	4.8	0.42	1	4.6	0.22	0	1.8	0.0	0.0
Malignant neoplasms	1	3.3	0.30	4	3.9	1.03	1	1.1	0.91	0	1.0	0.0	0	0.4	0.0	0.0
Respiratory tract	0	0.8	0.0	1	0.9	1.11	0	0.2	0.0	0	0.3	0.0	0	1.0	0.0	0.0
Leukemia, aleukemia	0	0.3	0.0	1	0.4	2.50	1	0.1	10.00	0	0.1	0.0	0	0.0	0.0	0.0
Diseases of cardiovascular system	3	5.5	0.55	3	6.3	0.48	1	1.7	0.59	0	2.0	0.0	0	0.6	0.0	0.0
Chronic nephritis, other renal	0	0.2	0.0	0	0.2	0.0	0	0.1	0.0	0	0.1	0.0	0	0.0	0.0	0.0
Cirrhosis of the liver	1	1.1	0.91	0	1.3	0.0	0	0.4	0.0	1	0.3	3.33	0	0.1	0.0	0.0
Total deaths, all causes ^c	18	31.0	0.58	23	37.1	0.62	3	9.6	0.31	1	7.1	0.14	1	4.2	0.24	0.24

Table 3 (contd.)

Underlying cause of death	Innocuous chemical and control, exclusively (N = 106)				No test (N = 1,719)				Total experience (N = 6,620)			
	Obs.	Exp.	O/E		Obs.	Exp.	O/E		Obs.	Exp.	O/E	
All trauma ^b	1	1.7	0.59		31	33.5	0.93		86	128.2	0.67	
Accidents	1	1.0	1.00		20	21.1	0.95		54	80.6	0.67	
Homicide	0	0.3	0.0		4	5.4	0.74		15	20.5	0.73	
Suicide	0	0.3	0.0		2	6.2	0.32		5	24.0	0.21	
All diseases	2	1.2	1.67		43	42.4	1.01		105	146.9	0.71	
Malignant neoplasms	2	0.3	6.67		6	9.6	0.63		30	32.9	0.91	
Respiratory tract	1	0.0	--d		3	2.6	1.15		10	8.5	1.18	
Leukemia, leukemia	1	0.0	--d		0	0.8	0.0		4	2.8	1.43	
Diseases of cardiovascular system	0	0.4	0.0		20	17.3	1.16		41	58.0	0.71	
Chronic nephritis, other renal	0	0.0	--		3	0.5	6.00		3	1.7	1.76	
Cirrhosis of the liver	0	0.1	0.0		3	2.9	1.03		7	10.5	0.67	
Total deaths, all causes ^c	3	2.9	1.03		84	75.8	1.11		222	275.1	0.81	

^aOnly: Men received this classification of chemical and none other, except for innocuous and control substances.

^bNot only: Men received this classification of chemical and one or more other experimental chemicals.

^cIncludes injury undetermined, whether accidentally or purposely inflicted, and injury resulting from operations of war.

^dIncludes the causes of death listed in this table plus all other causes, including cause not yet determined.

^eIndeterminate, because of zero denominator.

Table 4

Observed and Expected Deaths among Test Subjects
by Chemical Administered

Chemical	No. Subjects	No. Deaths		
		Observed	Expected	O/E
Anticholinesterase only	507	21	29.2	0.72
Sarin only	149	9	11.3	0.80
VX only	281	12	14.5	0.83
Remainder of group	77	0	3.3	0.0
Anticholinergic only	570	19	18.0	1.06
BZ only	136	6	6.9	0.87
EA 3443 only	28	1	1.1	0.91
EA 3580 only	45	0	1.4	0.0
Scopolamine only	85	5	2.0	2.50
Atropine only	62	3	1.7	1.76
EA 3834 only	64	1	1.2	0.83
Remainder of group	150	3	3.8	0.79
Irritant only	885	18	31.0	0.58
Mustard only	68	3	3.0	1.00
Remainder of group	817	15	28.0	0.54

REFERENCE

1. Keehn, R. J., 1980: Follow-up Studies of World War II and Korean Conflict Prisoners. III. Mortality to 1 January 1976, Am. J. Epidemiol. 111:194-211.